CDC
CENTERS FOR DISEASE CONTROL AND PREVENTION

MMWR
MORBIDITY AND MORTALITY WEEKLY REPORT

Vol. 41 / No. SS-6

CDC Surveillance Summaries

November 20, 1992

Measles Surveillance — United States, 1991

Years of Potential Life Lost Before Age 65, by Race, Hispanic Origin, and Sex — United States, 1986–1988

Group B Streptococcal Disease in the United States, 1990: Report from a Multistate Active Surveillance System

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Centers for Disease Control
and Prevention (CDC)
Atlanta, Georgia 30333

The *MMWR* series of publications is published by the Epidemiology Program Office, Centers for Disease Control and Prevention (CDC), Public Health Service, U.S. Department of Health and Human Services, Atlanta, Georgia 30333.

SUGGESTED CITATION

General: Centers for Disease Control and Prevention. CDC *Surveillance Summaries,* November 20, 1992. *MMWR* 1992;41:(No. SS-6).

Specific: Centers for Disease Control and Prevention. [Title of particular article.] In: CDC *Surveillance Summaries,* November 20, 1992. *MMWR* 1992; 41(No. SS-6): [inclusive page numbers].

Copies can be purchased from Superintendent of Documents, U.S. Government Printing Office, Washington, D.C. 20402-9325. Telephone: (202) 783-3238.

Contents

Most Recent Reports Published in the MMWR Surveillance Summaries

Subject	Responsible CIO*	Most Recent Report
Abortion	NCCDPHP	1992; Vol. 41, No. SS-5
AIDS/HIV		
Distribution by Racial/Ethnic Group	NCID	1988; Vol. 37, No. SS-3
Among Black and Hispanic Children and Women of Childbearing Age	NCEHIC	1990; Vol. 39, No. SS-3
Behavioral Risk Factors	NCCDPHP	1991; Vol. 40, No. SS-4
Birth Defects		
B.D. Monitoring Program (see also Malformations)	NCEHIC	1990; Vol. 39, No. SS-4
Contribution of B.D. to Infant Mortality Among Minority Groups	NCEHIC	1990; Vol. 39, No. SS-3
Breast and Cervical Cancer	NCCDPHP	1992; Vol. 41, No. SS-2
Campylobacter	NCID	1988; Vol. 37, No. SS-2
Chancroid	NCPS	1992; Vol. 41, No. SS-3
Cholera	NCID	1992; Vol. 41, No. SS-1
Coal Workers' Health (see also Mining)	NIOSH	1985; Vol. 34, No. 1SS
Congenital Malformations, Minority Groups	NCEHIC	1988; Vol. 37, No. SS-3
Contraception Practices	NCCDPHP	1992; Vol. 41, No. SS-4
Cytomegalovirus Disease, Congenital	NCID	1992; Vol. 41, No. SS-2
Dengue	NCID	1985; Vol. 34, No. 2SS
Dental Caries and Periodontal Disease Among Mexican-American Children	NCPS	1988; Vol. 37, No. SS-3
Dracunculiasis	NCID	1992; Vol. 41, No. SS-1
Ectopic Pregnancy	NCCDPHP	1990; Vol. 39, No. SS-4
Ectopic Pregnancy, Mortality	NCCDPHP	1987; Vol. 36, No. SS-2
Elderly, Hospitalizations Among	NCCDPHP	1991; Vol. 40, No. SS-1
Endometrial and Ovarian Cancers	EPO, NCCDPHP	1986; Vol. 35, No. 2SS
Escherichia coli O157	NCID	1991; Vol. 40, No. SS-1
Evacuation Camps	EPO	1992; Vol. 41, No. SS-4
Foodborne Disease	NCID	1990; Vol. 39, No. SS-1
Gonococcal Infection	NCPS, NCID	1984; Vol. 33, No. 4SS
Gonorrhea and Salpingitis, Teenagers	NCPS, NCID	1983; Vol. 32, No. 3SS
Health Surveillance Systems	IHPO	1992; Vol. 41, No. SS-4
Hepatitis	NCID	1985; Vol. 34, No. 1SS
Hepatitis, Viral	NCID	1983; Vol. 32, No. 2SS
Homicide	NCEHIC	1992; Vol. 41, No. SS-3
Homicides, Black Males	NCEHIC	1988; Vol. 37, No. SS-1
Hysterectomy	NCCDPHP	1986; Vol. 35, No. 1SS
Infant Mortality (see also National Infant Mortality; Birth Defects; Postneonatal Mortality)	NCEHIC	1990; Vol. 39, No. SS-3
Influenza	NCID	1992; Vol. 41, No. SS-5
Injury		
Death Rates, Blacks and Whites	NCEHIC	1988; Vol. 37, No. SS-3
Drownings	NCEHIC	1988; Vol. 37, No. SS-1
Falls, Deaths	NCEHIC	1988; Vol. 37, No. SS-1
Firearm-Related Deaths, Unintentional	NCEHIC	1988; Vol. 37, No. SS-1
In Developing Countries	NCEHIC	1992; Vol. 41, No. SS-1
In the Home, Persons <15 Years of Age	NCEHIC	1988; Vol. 37, No. SS-1
Motor Vehicle-Related Deaths	NCEHIC	1988; Vol. 37, No. SS-1
Objectives of Injury Control, State and Local	NCEHIC	1988; Vol. 37, No. SS-1
Objectives of Injury Control, National	NCEHIC	1988; Vol. 37, No. SS-1
Residential Fires, Deaths	NCEHIC	1988; Vol. 37, No. SS-1
Tap Water Scalds	NCEHIC	1988; Vol. 37, No. SS-1
Lead Poisoning, Childhood	NCEHIC	1990; Vol. 39, No. SS-4
Low Birth Weight	NCCDPHP	1990; Vol. 39, No. SS-3

*All abbreviations are listed at end of inventory. Readers should check individual summaries when more than one CIO is responsible.

Most Recent Reports Published in the MMWR Surveillance Summaries — Continued

Subject	Responsible CIO*	Most Recent Report
Malaria, Imported	NCID	1983; Vol. 32, No. 3SS
Malformations (see also Birth Defects)	NCEHIC	1985; Vol. 34, No. 2SS
Maternal Mortality	NCCDPHP	1991; Vol. 40, No. SS-2
Measles	NCPS	1992; Vol. 41, No. SS-6
Mining (see also Coal Workers' Health)	NIOSH	1986; Vol. 35, No. 2SS
National Infant Mortality (see also Infant Mortality; Birth Defects)	NCCDPHP	1989; Vol. 38, No. SS-3
Nosocomial Infection	NCID	1986; Vol. 35, No. 1SS
Occupational Injuries/Disease		
Among Loggers	NIOSH	1983; Vol. 32, No. 3SS
Hazards, Occupational	NIOSH	1985; Vol. 34, No. 2SS
In Meatpacking Industry	NIOSH	1985; Vol. 34, No. 1SS
State Activities	NIOSH	1987; Vol. 36, No. SS-2
Treated in Hospital Emergency Rooms	NIOSH	1983; Vol. 32, No. 2SS
Ovarian Cancer (see Endometrial and Ovarian Cancers)		
Parasites, Intestinal	NCID	1991; Vol. 40, No. SS-4
Pediatric Nutrition	NCCDPHP	1983; Vol. 32, No. 4SS
Pelvic Inflammatory Disease	NCPS	1983; Vol. 32, No. 4SS
Plague	NCID	1985; Vol. 34, No. 2SS
Plague, American Indians	NCID	1988; Vol. 37, No. SS-3
Pneumoconiosis, Coal Miners	NIOSH	1983; Vol. 32, No. 1SS
Poliomyelitis	NCPS	1992; Vol. 41, No. SS-1
Postneonatal Mortality	NCCDPHP	1991; Vol. 40, No. SS-2
Pregnancy, Teenage	NCCDPHP	1987; Vol. 36, No. 1SS
Psittacosis	NCID	1983; Vol. 32, No. 1SS
Rabies	NCID	1989; Vol. 38, No. SS-1
Racial/Ethnic Minority Groups	Various	1990; Vol. 39, No. SS-3
Respiratory Disease	NCEHIC	1992; Vol. 41, No. SS-4
Reye Syndrome	NCID	1984; Vol. 33, No. 3SS
Rocky Mountain Spotted Fever	NCID	1984; Vol. 33, No. 3SS
Rotavirus	NCID	1992; Vol. 41, No. SS-3
Rubella and Congenital Rubella	NCPS	1984; Vol. 33, No. 4SS
Salmonella	NCID	1988; Vol. 37, No. SS-2
Salpingitis (see Gonorrhea and Salpingitis)		
Sexually Transmitted Diseases in Italy	NCPS	1992; Vol. 41, No. SS-1
Smoking	NCCDPHP	1990; Vol. 39, No. SS-3
Streptococcal Disease (Group B)	NCID	1992; Vol. 41, No. SS-6
Sudden Unexplained Death Syndrome Among Southeast Asian Refugees	NCEHIC, NCPS	1987; Vol. 36, No. 1SS
Suicides, Persons 15–24 Years of Age	NCEHIC	1988; Vol. 37, No. SS-1
Summer Mortality	NCEHIC	1983; Vol. 32, No. 1SS
Syphilis	NCPS	1991; Vol. 40, No. SS-3
Toxic-Shock Syndrome	NCID	1984; Vol. 33, No. 3SS
Trichinosis	NCID	1991; Vol. 40, No. SS-3
Tubal Sterilization Among Women	NCCDPHP	1983; Vol. 32, No. 3SS
Tuberculosis	NCPS	1991; Vol. 40, No. SS-3
Water-Related Disease	NCID	1991; Vol. 40, No. SS-3
Years of Potential Life Lost	EPO	1992; Vol. 41, No. SS-6

Abbreviations

NCCDPHP	National Center for Chronic Disease Prevention and Health Promotion
NCEHIC	National Center for Environmental Health and Injury Control
NCID	National Center for Infectious Diseases
CIO	Centers/Institute/Offices
NCPS	National Center for Prevention Services
IHPO	International Health Program Office
EPO	Epidemiology Program Office
NIOSH	National Institute for Occupational Safety and Health

Measles Surveillance — United States, 1991

William L. Atkinson, M.D., M.P.H.
Stephen C. Hadler, M.D.
Susan B. Redd
Walter A. Orenstein, M.D.
Division of Immunization
National Center for Prevention Services

Summary

A total of 9,643 measles cases was reported from the United States in 1991, a 65.3% decrease from the 27,786 cases reported in 1990. The overall incidence of measles was 3.9 cases per 100,000 population. The highest age-specific incidence was among children <12 months of age (46.9/100,000) and 1–4 years of age (19.6/100,000). Incidence rates among American Indians, Hispanic, and black children <5 years of age were 19, 6, and 4 times that for non-Hispanic white children, respectively. More than 61% of all cases were reported from seven large outbreaks, which involved predominantly unvaccinated preschool-age children in large urban areas. Although reported measles cases decreased in 1991 compared with 1989–1990, only a sustained effort to provide age-appropriate vaccination will prevent another resurgence of measles.

INTRODUCTION

After almost a decade of relatively few reported cases, a major resurgence of measles occurred in the United States during the period 1989–1990. The number of reported cases declined in 1991, but remained above the annual number reported during most of the 1980s. This report describes the surveillance and epidemiology of reported cases of measles in 1991, contrasts these cases with those reported in the previous 2 years, and discusses the implications of the findings.

METHODS

To characterize measles in 1991, cases of measles reported to the National Notifiable Diseases Surveillance System (NNDSS) of CDC were reviewed. For purposes of disease surveillance, a clinical case of measles is defined as an illness consisting of a generalized maculopapular rash lasting ≥3 days, fever 38.3 C (101 F), if measured, and the occurrence of cough, coryza, or conjunctivitis. A confirmed case of measles is defined as one that meets the clinical case definition and is either serologically confirmed or epidemiologically linked to another clinical case (*1*). Only confirmed cases reported to NNDSS were included in this analysis. Information reported to NNDSS includes age, county of residence, race/ethnicity, and date of onset of illness. These data were used to calculate overall unadjusted and age- and race-specific incidences, based on 1990 Census data. For race-specific incidence, only cases from states that reported race and ethnicity for at least 66% of all cases were used.

The Division of Immunization, National Center for Prevention Services, CDC, collects supplemental information on reported measles cases. Information collected

includes vaccination history, setting of transmission, complications, hospitalizations, serologic data, and whether a case occurred as part of an outbreak (defined as the occurrence of five or more epidemiologically related cases). On the basis of investigations of reported measles cases, state health departments determine the most likely setting of exposure or transmission. Measles outbreaks are classified as predominantly preschool-age, school-age, or postschool-age, i.e., children <5 years of age, children and teenagers from 5 to 19 years of age, or adults ≥20 years of age, respectively (*2*).

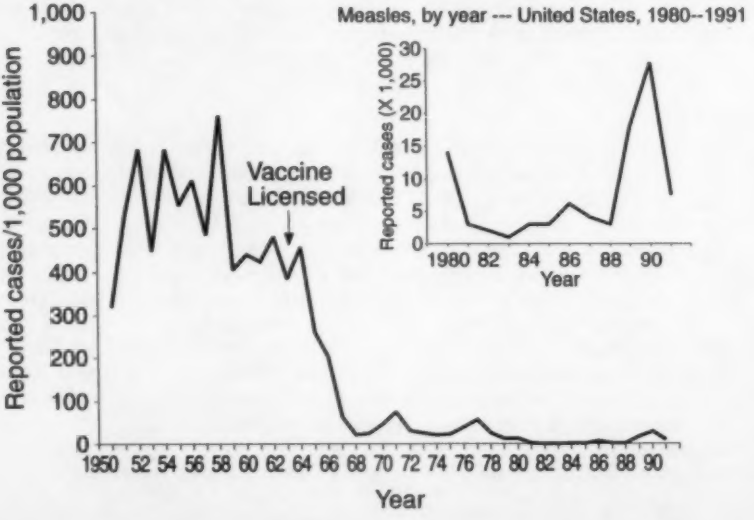
Information on measles-associated deaths is collected by the Division of Immunization through active surveillance and is provisional. An attempt is made to verify that deaths are related to measles by obtaining clinical records when available (e.g., hospital records, death certificates, autopsy reports). The final total of measles-associated deaths is reported by the National Center for Health Statistics; these data will not be available for approximately 2 years.

RESULTS

Local and state health departments reported a total of 9,643 measles cases for 1991 — a 65.3% decrease from the 27,786 cases reported for 1990 (*3*) (Figure 1). Cases were reported from 42 states and 372 (11.9%) counties, compared with 49 states and 782 (24.9%) counties in 1990 (*4*). Thirty-six suspected measles-associated deaths have been reported for 1991.

Eleven states reported at least 100 cases each. Four states reported >1,000 cases each, accounting for 6,831 (70.8%) of the total reported cases: New York (2,306, including 1,909 cases reported from New York City), California (1,959), Pennsylvania (1,448), and New Jersey (1,118). Incidence greater than 4.0/100,000 population occurred in

FIGURE 1. Reported measles (rubeola) cases, by year — United States, 1950–1991



Idaho (46.2), New Jersey (14.5), Utah (13.0), New York (12.8), Pennsylvania (12.2), Arizona (12.0), New Mexico (7.5), and California (6.6).

The median age of persons reported with measles was 5.2 years in 1991, 5.7 years in 1990, and 12.0 years in 1989. Children <5 years of age accounted for 49.3% of measles cases, compared with 48.6% of cases in 1990 (Table 1). Persons ≥20 years of age accounted for 19.6% of all reported cases in 1991, compared with 22.3% in 1990.

The overall incidence in 1991 was 3.9 cases per 100,000 population. Estimated age-specific incidence was lower in 1991 than in 1990 for all age groups. As in previous years, the highest incidence was among children <1 year of age (46.9/100,000) and 1–4 years (19.6/100,000) (Table 1). Estimated incidence for 3-month age groups of children <2 years of age indicated that the risk of measles was highest among those 10–12 months of age (91.6/100,000), 7–9 months of age (74.2/100,000), and 13–15 months of age (65.3/100,000), with lower incidence in children <6 months of age (41.7/100,000) and 16–23 months of age (30.3/100,000).

Information on race and ethnicity was available for 5,751 (59.5%) cases reported from 23 states.* Of these 5,751 cases, 2,461 (42.8%) occurred among non-Hispanic whites, 902 (15.7%) occurred among non-Hispanic blacks, 1,956 (34.0%) occurred among Hispanics, 338 (5.9%) occurred among American Indians, and 94 (1.6%) occurred among other racial or ethnic groups. Measles incidence in these states was highest for American Indians (35.4/100,000), Hispanics (10.9/100,000 population), and non-Hispanic blacks (5.9/100,000) and was lowest for non-Hispanic whites (2.6/100,000). Among children <5 years of age, the incidence of measles for American Indian, Hispanic, and black children was 19, 6, and 4 times that for non-Hispanic white children, respectively (Figure 2).

Setting of transmission was reported for 3,361 (34.9%) cases. The most frequently reported sites of transmission were home (1,378 cases, 41.0%), school (622 cases, 18.5%), medical settings — including hospital wards, emergency departments, and

*Alabama, Alaska, Arizona, Arkansas, California, Delaware, Florida, Hawaii, Idaho, Kansas, Maine, Massachusetts, Nebraska, Nevada, New York (including New York City), North Carolina, Oregon, South Carolina, Tennessee, Texas, Utah, Virginia, and Washington.

TABLE 1. Age distribution and estimated incidence* of measles — United States, 1990 and 1991

Age group (years)	1990			1991			
	No.	(%)	Rate†	No.	(%)	Rate†	% Change
<1	4,709	(16.9)	119.3	1,852	(19.2)	46.9	-68.0%
1–4	8,783	(31.5)	59.3	2,904	(30.1)	19.6	-66.2%
5–9	2,687	(9.6)	14.9	991	(10.2)	5.5	-62.8%
10–14	2,278	(8.2)	13.4	905	(9.4)	5.3	-60.2%
15–19	3,118	(11.2)	17.4	1,102	(11.4)	6.2	-64.8%
20–24	2,550	(9.1)	13.3	660	(6.8)	3.5	-73.9%
≥25	3,660	(13.1)	2.3	1,230	(12.8)	0.8	-65.2%
Total	**27,786**	**(100.0)**	**11.2**	**9,643**	**(100.0)**	**3.9**	**-65.2%**

*Cases per 100,000 population.
†Rates were calculated based on the assumption that the age distributions of 108 reported cases of unknown age in 1990 and 50 of unknown age in 1991 were similar to the distribution of cases of known age.

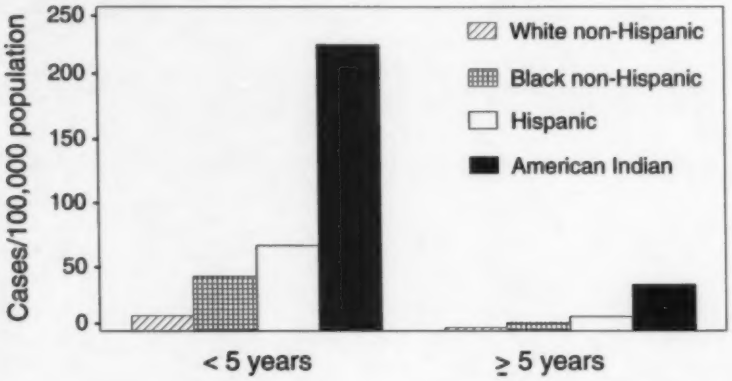
physicians' offices (617 cases, 18.4%) — and day care facilities (147 cases, 4.4%). Setting of transmission varied by age. Among those cases with known setting of transmission, the most frequently reported sites for children <5 years of age were home (726, 52.2%) and medical settings (359, 25.8%); among persons 5–19 years of age, school (579, 45.7%) and home (455, 35.9%) were most common; and among persons ≥20 years of age, home (197, 28.1%) and medical settings (192, 27.4%) were most often reported.

Persons presumably infected abroad accounted for 87 (0.9%) cases; an additional 65 cases were epidemiologically linked within two generations to imported cases. The country of origin is known for 69 international importations. Of these, 11 (15.9%) were acquired in Mexico, 15 (21.7%) in other Central American, South American, or Caribbean countries, including Puerto Rico, and six (8.7%) in Canada. The remaining 37 importations with known country of origin were acquired in Asia (16, including 10 in Japan), Europe (11), Oceania (8), and Africa (2).

OUTBREAKS

The 11 largest measles outbreaks in 1991 (Table 2) ranged from 108 to 1,909 cases and accounted for 6,793 (70.4%) reported cases. Seven of these large outbreaks involved predominantly preschool-age children and accounted for 5,934 (61.5%) cases. The largest outbreaks occurred mostly among preschool-age children in New York City (1,909 cases), Philadelphia (1,338 cases), Los Angeles (1,166 cases), and Newark-Jersey City-Elizabeth, New Jersey (824 cases). In Los Angeles, >12,000 cases of measles have been reported from the four-county area (Los Angeles, Orange, San Bernadino,

FIGURE 2. Measles incidence, by age and race — United States, 1991*



*Data shown for 23 states that reported race for >66% of cases.

TABLE 2. Measles outbreaks with >100 reported cases — United States, 1991

State	Location	Date of first and last case	1991 cases	Type*	Number (%) < 5 years	Number (%) unvaccinated
New York	Bronx, Kings, New York (Manhattan), Queens, Richmond Counties	12/88–†	1,909	Preschool	1,192 (62.4)	1,676 (87.8)
Pennsylvania	Philadelphia, Montgomery, Bucks, Chester, Delaware Counties	10/90–6/91	1,338	Preschool	632 (47.2)	1,079 (80.6)
California	Los Angeles, Orange, Riverside, San Bernadino Counties	8/87–5/92	1,166	Preschool	547 (46.9)	946 (81.1)
New Jersey	Essex, Hudson, Union, Passaic, Middlesex, Bergen Counties	12/90–9/91	824	Preschool	533 (64.7)	649 (78.8)
Idaho	Fremont, Madison, Ada, Canyon, Bannock, Latah Counties§	2/91–9/91	465	School	91 (19.6)	361 (77.6)
Arizona	Navajo, Coconino, Mohave, Apache, Yavapai Counties	12/90–8/91	383	Preschool	220 (57.4)	325 (84.9)
Florida	Duval, Nassau, St. Johns, Clay Counties§	4/91–1/92	176	Preschool	127 (72.2)	143 (81.3)
Utah	Davis, Rich, Iron, Weber Counties	3/91–8/91	149	School	45 (30.2)	79 (53.0)
New Jersey	Camden County	11/90–7/91	138	Preschool	98 (71.0)	112 (81.2)
New York	Suffolk County	12/90–7/91	137	Postschool	47 (34.3)	116 (84.7)
Maryland	Anne Arundel, Calvert, Cecil, Baltimore, Howard Counties§	2/91–5/91	108	School	18 (16.7)	85 (79)

* Predominant age group affected. See text for description.
† Cases continue to be reported.
§ Other counties reported cases in addition to those counties listed.

and Riverside Counties) since transmission began in August 1987; transmission peaked in 1990, with >7,500 cases reported. Cases decreased substantially in 1991. An outbreak involving predominantly preschool children in Arizona began in December 1990 on the Navajo Reservation and spread to at least four other counties. The outbreak in Philadelphia, involving predominantly preschool-age children, occurred between October 1990 and June 1991. However, within this outbreak, a second outbreak occurred that involved members of a religious group who do not accept conventional medical care, including vaccination. This outbreak accounted for approximately 480 cases and six deaths. Case-persons reported from the outbreak among the religious group were older than those reported elsewhere in Philadelphia; the median age among ill members of the religious group was 96 months, compared with 31 months overall in the Philadelphia cases not associated with the religious group.

Large outbreaks involving predominantly school-age children in Utah, Idaho, and Maryland accounted for 722 (7.5%) cases. In Utah, schools were the predominant setting for an outbreak of 149 cases, which began in Davis County and spread to three other counties. In Idaho, a measles outbreak began in a college in Fremont County and eventually spread to 19 other counties (for a total of 465 cases). Much of the measles transmission occurred via attendance at high school wrestling matches and other school activities. Similarly, high school wrestling tournaments facilitated transmission of measles in Maryland: 108 cases occurred in 10 counties subsequent to an initial case in a high school student in Howard County. In these three outbreaks, 47%, 22%, and 21% of cases had documentation of measles vaccination, respectively.

A large outbreak involving predominantly postschool-age persons (≥20 years of age) in Suffolk County, New York, was probably linked to the outbreak in New York City. Most adults reported in this outbreak acquired measles in a Central American migrant community, a homeless shelter, and a hospital. Thirty-four percent of cases from this outbreak were among children <5 years of age.

VACCINATION STATUS

Vaccination status is known for 8,534 (88.5%) reported cases. Persons with unknown vaccination status were assumed to be unvaccinated. A total of 1,966 (20.4%) persons was appropriately vaccinated (one dose of measles vaccine on or after the first birthday) (Table 3), including 14 persons who had received two doses of measles vaccine. Approximately 70% of appropriately vaccinated persons with measles were 5–19 years of age. The remaining 7,677 (79.6%) persons with measles were unvaccinated or inadequately vaccinated (i.e., vaccinated before their first birthday). Of these persons, routine vaccination was indicated* for 3,680 (38.2% of total). Thirty-eight percent of these vaccine-eligible persons were children 16 months to 4 years of age, 29% were school-age children 5–19 years of age, and 34% were adults ≥20 years of age.

Measles occurred in 3,256 (33.8% of total) persons for whom routine vaccination was not indicated, of whom 2,800 (86.0%) were children <16 months of age. Children

*Unvaccinated persons <16 months of age without medical contraindications or religious exemption to vaccination. This represents a minimal estimate, since the Advisory Committee on Immunization Practices (ACIP) recommends that the routine age for the first dose of measles vaccine be lowered from 15 months to 12 months in areas where preschool-age children are at high risk of measles (*5*).

<16 months of age accounted for 29.3% of all reported cases in 1991. In contrast, this group accounted for 26.3% of reported cases in 1990 and 18.6% in 1989. Nine hundred forty-nine (33.6%) cases reported among children <16 months of age were ages 12–15 months and may have been eligible for vaccination, depending on whether they lived in a county where preschool-age children are at high risk of measles (*5*).

Seven hundred forty-one (7.7% of total) persons were unvaccinated for other reasons; 672 (90.7%) of these were persons with religious or philosophic exemptions to vaccination.

COMPLICATIONS OF MEASLES

One or more complications were reported for 2,684 (27.8%) cases, including diarrhea in 1,194 (12.4%), otitis media in 1,308 (13.6%), pneumonia in 815 (8.5%), and encephalitis in 13 (0.1%).* Hospitalization was reported for 2,549 (26.4%) persons, for a total of at least 12,578 days (median, 4 days; range, 1–180 days). As in previous years (*6*), complications and hospitalization were more frequent among children <5 years and adults ≥20 years of age (Table 4).

DEATHS

A provisional total of 36 measles-associated deaths was reported, for a case-fatality rate of 3.7 deaths per 1,000 reported cases. Deaths were reported from six states: New York (15), Pennsylvania (8), California (8), New Jersey (3), Arizona (1), and Florida (1). Thirteen (36.1%) deaths occurred among children <5 years of age, including nine (25.0%) <12 months of age, and 12 (33.3%) deaths occurred among children 5–19 years of age. Six of the deaths among children 5–19 years of age occurred among members of a religious group in Philadelphia who do not accept conventional medical care, in-

* Some persons with measles were reported to have more than one complication.

TABLE 3. Classification of measles cases — United States, 1991*

Classification	No.	% of total
Unvaccinated	7,677	79.6
Vaccine indicated	3,680	38.2
Vaccine not routinely indicated	3,256	33.8
Persons <16 months of age	2,800	29.0
Persons born before 1957	418	4.3
Laboratory immunity/physician diagnosis	4	0.0
Medical exemption	34	0.4
Other	741	7.7
Non-U.S. citizen	69	0.7
Religious/philosophic exemption	672	7.0
Appropriately vaccinated†	1,966	20.4
Total	**9,643**	**100.0**

*Vaccination status available for 8,534 (88.5%) of reported cases. Persons with unknown vaccination status were assumed to be unvaccinated, except children <16 months of age, who were classified as unvaccinated and vaccine not routinely indicated.
†One dose of measles vaccine on or after the first birthday.

cluding vaccination. The remaining 11 (30.6%) deaths occurred in adults aged ≥20 years. Only two (5.4%) persons who died were known to have been vaccinated. At least five persons who died were known to have been infected with human immunodeficiency virus.

DISCUSSION

The 9,643 measles cases reported in 1991 represent the third year of a resurgence of measles in the United States, which began in 1989 and peaked in 1990. The decreasing incidence of measles appears to be continuing in 1992, with only 1,996 cases reported as of September 19, compared with 8,764 cases reported during the same period in 1991 (*7*).

Although the number of reported measles cases decreased in 1991 compared with 1990, the characteristics of reported cases are similar to those reported in 1990. The change in age distribution of measles cases first noted in 1989 (*8*) and 1990 (*4*) continued in 1991. In 1990, the proportion of cases among children <5 years of age exceeded the proportion in school-age children for the first time since detailed information on the age of reported case-persons became available in 1973. The proportion of cases among children <5 years of age in 1991 (49.3%) was slightly higher than in 1990 (48.6%). Furthermore, the proportion of cases among children <1 year of age continued to increase, constituting 19.2% of all reported cases in 1991, compared with 16.9% in 1990 (Figure 3) and a median of only 8.1% of reported cases in 1980–1988. Children 12–15 months of age, an age group eligible for vaccination in areas where preschool-age children are at high risk for measles, accounted for 10% of all reported cases, up from a median of 6.7% from 1985 to 1989. The cause of the increase in measles cases among children <15 months of age is currently under investigation; possible contributing factors include increased exposure to measles from older, unvaccinated children with measles and earlier susceptibility to measles due to transplacental transfer of lesser amounts of measles antibody from young mothers whose measles immunity is from vaccine rather than wild measles virus (*9–12*).

As in the period 1989–1990 (*4,8*), the incidence of measles was greater for non-Hispanic black and Hispanic children <5 years of age than for non-Hispanic white children. Unlike previous years, American Indian children had the highest incidence of measles. The majority of cases among American Indian children in 1991 occurred in a single large outbreak in Arizona. Almost 30% of cases in this outbreak occurred among children <12 months of age.

TABLE 4. Proportion of reported measles with complications, by age group — United States, 1991

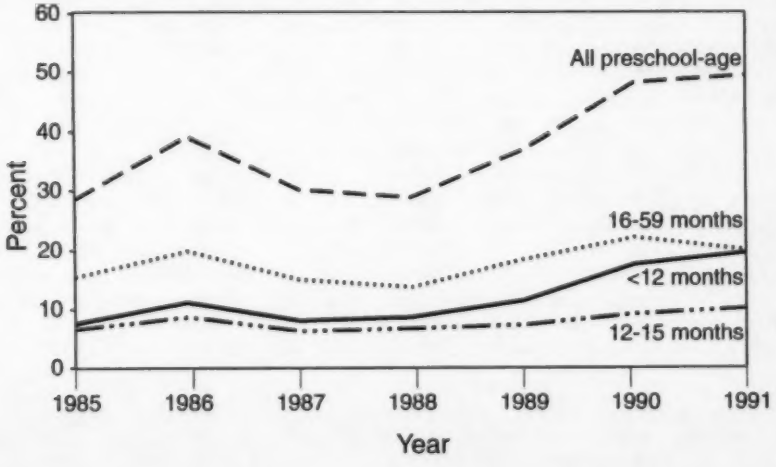
	Age group							
	< 5 years		5–19 years		≥ 20 years		Total	
Condition	No.	(%)	No.	(%)	No.	(%)	No.	(%)
Otitis media	1,105	(23)	138	(5)	65	(3)	1,308	(14)
Diarrhea	740	(16)	212	(7)	242	(13)	1,194	(12)
Pneumonia	502	(11)	97	(3)	216	(11)	815	(8)
Encephalitis	3	(0.1)	3	(0.1)	7	(0.4)	13	(0.1)
Hospitalization	1,525	(32)	343	(11)	681	(36)	2,549	(26)
Total cases	**4,756**		**2,998**		**1,890**		**9,643**	

Transmission in medical settings (hospitals, emergency rooms, and physicians' offices) accounted for 617 reported measles cases in 1991, which was more than 6% of all reported cases. This is the highest proportion of reported measles infection in these settings since detailed information on sites of transmission became available in 1985 (*13*; CDC unpublished data). Medical settings were particularly important for preschool-age children and adults ≥20 years, accounting for 26% and 27% of all cases with known setting of transmission in these age groups, respectively. Data on the occupation of adults with measles acquired in medical settings in 1991 are not yet available. However, previous studies have shown that most adults who acquire measles in medical settings are health-care workers and most are unvaccinated (*13*).

The pattern of measles outbreaks in 1991 was similar to that observed in 1989 and 1990 (*4,8*), with the largest outbreaks occurring among preschool-age children. These outbreaks are the largest contributor to the overall change in the age distribution and the decreasing median age of reported measles cases. The major cause for these outbreaks has been the lack of timely measles vaccination of preschool-age children in inner cities (*14,15*). Collaborative efforts of CDC's Division of Immunization, other federal agencies, state and local health departments, and the private sector are now under way to improve vaccine coverage in these areas.

Although the largest outbreaks of measles were among preschool-age children, outbreaks continue to occur among school-age children, which may lead to widespread transmission of measles throughout a state. The measles outbreaks in Utah, Idaho, and Maryland in 1991 emphasize this point. The proportion of persons who became ill with measles despite a documented history of vaccination was lower than those previously reported among school-age persons (*2*). However, many persons could not provide documentation of vaccination and were considered unvaccinated.

FIGURE 3. Proportion of total measles cases by age group — United States, 1985–1991



Because the majority of persons in these outbreaks entered school after state immunization laws were enacted, it is likely that most had been vaccinated, but records were not available for verification. As a result of widespread transmission of measles among older school-age persons in 1991, and to address the issue of lack of documentation of vaccination, Idaho has revised its state immunization law to require documentation of vaccination of children at all grade levels instead of only children in kindergarten through fifth grades.

The provisional total of 36 measles-associated deaths in 1991 brings to a total of 166 deaths reported to CDC since the resurgence of measles began in 1989. As in previous years, most deaths were reported from areas with large outbreaks among preschool-age children (*4,8*). However, the age distribution of deaths in 1991 is notable for a greater proportion of deaths among older age groups. During the period 1989–1990, 60% of all suspected measles-associated deaths were among children <5 years of age. This age group accounted for 36% of deaths in 1991. In contrast, 12% of all reported deaths during the period 1989–1990 were among persons 5–19 years of age, compared with 32% in 1991. This change in distribution of measles deaths in 1991 is partially due to the six persons who died among the religious group in Philadelphia, all of whom were ages 5–14 years. Persons ≥20 years of age accounted for 28% of all deaths during the period 1989–1991 and 20% of reported cases. As in 1989 and 1990, almost all persons who died as a result of measles were unvaccinated.

In 1991, 20.4% of all reported persons had been appropriately vaccinated, comparable with 1990 rates. The most notable change in vaccination status, however, was the increasing proportion of persons younger than the routine age of vaccination (16 months). Many of these children could have been protected from measles. The ACIP recommends that in areas where preschool-age children are at high risk, measles vaccine be administered at 12 months of age (*5*).*

Because of recent outbreaks of measles among preschool-age children, many counties in the United States currently qualify as at high risk for measles. In addition, many metropolitan areas of the United States have large inner-city populations, and measles vaccine coverage is known to be suboptimal in all such areas that have been studied (*15*). Therefore, most metropolitan areas in the United States should strongly consider revising their policies in accordance with current ACIP and American Academy of Pediatrics (*16*) recommendations, which call for administration of measles vaccine to children at 12 months of age in high-risk areas.

The causes of the resurgence of measles during the years 1989–1991 are not known with certainty. Contributing factors likely include low measles vaccine coverage levels among preschool-age children, particularly those living in inner-city areas; earlier development of measles susceptibility among children <15 months of age; and the occurrence of measles epidemics throughout North and Central America during this interval (*17*). There is no evidence that measles vaccine efficacy has decreased. A recent vaccine efficacy study among preschool-age children in California determined a single dose to be 95% effective in preventing measles among this age group (*18*).

*A county with more than five cases of measles among preschool-age children during each of the past 5 years, a county with a recent outbreak among unvaccinated preschool-age children, or a county with a large inner-city urban population.

In addition, the causes for the current decrease in measles are not clear. It is unlikely that all susceptible preschool-age children have been infected, even in cities with the highest incidence of measles. For example, measles vaccine coverage for children 2 years of age in the United States is estimated to be approximately 70%–80% (*19*). Thus, in one age group (12–23 months of age), there would be approximately 800,000–1,200,000 susceptible children, assuming a birth cohort of 4 million children. In 1990, there were 4,735 cases reported among children 12–23 months of age, and in 1991 only 1,703 cases. These reported cases did not substantially reduce overall susceptibility in this age group. A greater reduction in susceptibility resulted from the administration of almost 400,000 more doses of measles vaccine to 1-year-old children in the public sector in 1991 compared with 1988 (*20*). However, the precise role of this increased vaccination in curtailing transmission is not known with certainty.

Whatever the cause of the current decrease in measles in the United States, it is critical that efforts continue to improve age-appropriate vaccination coverage, particularly among preschool-age children living in inner-city areas. Improvements in vaccine coverage among preschool-age children are essential for preventing another resurgence of measles.

Acknowledgment

The authors thank Immunization Program personnel in the 63 state and local United States immunization project areas for supplying data used in this analysis.

References

1. CDC. Classification of measles cases and categorization of measles elimination programs. MMWR 1982;31:707–11.
2. Markowitz LE, Preblud SR, Orenstein WA, et al. Patterns of transmission in measles outbreaks in the United States, 1985–1986. N Engl J Med 1989;320:75–81.
3. CDC. Summary of notifiable diseases, United States, 1990. MMWR 1990;39(53).
4. CDC. Measles — United States, 1990. MMWR 1991;40:369–72.
5. ACIP. Measles prevention: recommendations of the Immunization Practices Advisory Committee. MMWR 1989;38(No. S-9).
6. Atkinson WL, Markowitz LE. Measles and measles vaccine. Semin Pediatr Infect Dis 1991;2:100–7.
7. CDC. Cases of selected notifiable diseases, United States, weeks ending September 19, 1992 and September 21, 1991 (28th week) [Table II]. MMWR 1992;41:712.
8. CDC. Measles — United States, 1989, and first 20 weeks 1990. MMWR 1990;39:353–5, 361–3.
9. Papania MJ, Lee S, Atkinson WL, et al. Risk factors for measles in exposed children <16 months old, New Jersey. Presented at the 41st annual Epidemic Intelligence Service Conference, Atlanta, Georgia, April 10, 1992.
10. Lennon JL, Black FL. Maternally derived measles immunity in era of vaccine-protected mothers. J Pediatr 1986;108:671–6.
11. Black FL, Berman LL, Borgono JM, et al. Geographic variation in infant loss of maternal measles antibody and in prevalence of rubella antibody. Am J Epidemiol 1986;124:442–52.
12. Pabst HF, Spady DW, Marusyk RG, et al. Reduced measles immunity in infants in a well-vaccinated population. Pediatr Infect Dis J 1992;11:525–9.
13. Atkinson WL, Markowitz LE, Adams NC, Seastrom GR. Transmission of measles in medical settings — United States, 1985–1989. Am J Med 1991;91(suppl 3B):320S–324S.
14. National Vaccine Advisory Committee. The measles epidemic: the problems, barriers, and recommendations. JAMA 1991;266:1547–52.
15. CDC. Retrospective assessment of vaccination coverage among school-aged children — selected U.S. cities, 1991. MMWR 1992;41:103–7.
16. American Academy of Pediatrics. Measles: reassessment of the current immunization policy. Pediatrics 1989;84:1110–3.

17. Atkinson WL, Orenstein WA, Krugman S. The resurgence of measles in the United States, 1989–1990. Annu Rev Med 1992;43:451–63.
18. King GE, Markowitz LE, Patriarca PA, Dales LG. Clinical efficacy of measles vaccine during the 1990 measles epidemic. Pediatr Infect Dis J 1991;10:883–8.
19. Bernier RH. Assessment of immunization coverage: a critical element in the strategy to reach 90% levels. In: 25th National Immunization Conference Proceedings, June 10–13, 1991, Washington, DC. Atlanta: CDC, 1992.
20. CDC. Public-sector vaccination efforts in response to the resurgence of measles among preschool-aged children — United States, 1989–1991. MMWR 1992;41:522–5.

Years of Potential Life Lost Before Age 65, by Race, Hispanic Origin, and Sex — United States, 1986–1988

Jean-Claude A. Desenclos, M.D.*
Robert A. Hahn, Ph.D., M.P.H.
Division of Surveillance and Epidemiology
Epidemiology Program Office

Summary

A substantial proportion of mortality among young persons is preventable. National vital statistics were used to establish a baseline for the surveillance of rates of years of potential life lost before age 65 (YPLL <65) in the United States. Rates of YPLL <65 were calculated for 1986 through 1988 for leading causes of preventable death, by race, Hispanic origin, and sex. U.S. racial and ethnic populations differed widely in YPLL <65. Among males, the rate (per 1,000 population <65 years) of YPLL <65 was highest for non-Hispanic blacks (140.0), followed by American Indians/Alaskan Natives (100.9), Hispanics (74.3), non-Hispanic whites (68.3), and Asians/Pacific Islanders (38.2). Among females, the rate was highest for non-Hispanic blacks (73.7), followed by American Indians/Alaskan Natives (52.0), non-Hispanic whites (35.7), Hispanics (32.9), and Asians/Pacific Islanders (23.2). For non-Hispanic blacks, the high rate of YPLL <65 was due to increased rates for all causes of death considered, particularly homicide. The high rate for American Indians/Alaskan Natives was due principally to deaths from four causes: unintentional injuries, cirrhosis, suicide, and diabetes. Asians/Pacific Islanders had low rates for most causes of death. In setting health-care priorities and prevention strategies to reduce the large racial-ethnic gap in early deaths, it is essential to recognize the differences in causes of premature mortality among sex, racial, and ethnic populations. Periodic reassessment of YPLL <65 among these groups provides a simple, timely, and representative means of conducting surveillance to measure the impact of intervention strategies on a national basis.

INTRODUCTION

A substantial proportion of mortality among young persons is potentially preventable and has been referred to as "premature" (*1,2*). Mortality statistics based on crude and adjusted death rates fail to assess the burden of deaths among younger persons, since these rates are dominated by deaths of the elderly (*2*). The measure of years of potential life lost before some selected age — most often 65 (YPLL <65) — gives a more accurate picture of premature mortality by weighting deaths occurring at younger ages more heavily than those occurring at older ages (*2*). Periodic assessment of YPLL <65 is a useful means of surveillance for setting priorities, guiding prevention program design, and planning the delivery of health-care services (*2–4*).

*Currently with the European Centre for the Epidemiological Monitoring of AIDS, Hopital National de Saint-Maurice, 14 rue du Val d'Osne, 94410 Saint-Maurice, France; telephone: (33) 1 43 96 65 45, FAX: (33) 1 43 96 50 81.

In the United States, YPLL <65 is tabulated annually for the 15 leading causes of death (*1,2*). Although a few studies have analyzed rates of YPLL <65 for whites and blacks (*2,5*), no system has been in place for comparing, on an ongoing and systematic basis, the relative importance of sex-specific premature mortality by race and Hispanic origin for leading causes of death. In this report, baseline surveillance data are provided for examination of rates of early death within sex, racial, and ethnic groups. Age-adjusted death rates and rates of YPLL <65 by race, Hispanic origin, and sex in the United States for 1986 through 1988 are analyzed.

MATERIALS AND METHODS

The CDC's National Center for Health Statistics Mortality Detail Data Tapes 1986–1988 (*6–8*) were used to calculate the mean annual number of deaths and mean YPLL <65 from 1986 through 1988. YPLL <65 was obtained by multiplying the number of deaths for each age group by the years of life lost before age 65 (difference between the 65-year end point and the midpoint of the age group) (*2*). For example, in the population 25–34 years old, the midpoint is 30 years and the YPLL <65 is 35 years times the number of deaths that occurred in the 25- to 34-year-old group. Age-specific YPLL <65 are then summed to give a YPLL <65 for selected causes of death and for all causes combined. For both males and females, YPLL <65 was computed for the following groups: non-Hispanic whites, non-Hispanic blacks, all Hispanics (black or white), specific Hispanic groups (i.e., Mexicans, Puerto Ricans, and Cubans), Native Americans (i.e., American Indians, Alaskan Natives, and Aleuts), and Asians/Pacific Islanders. Conditions were selected because of their high rates of mortality or their association with known, practical means of prevention; they included selected chronic diseases (*9*), homicide, suicide, unintentional injuries, drug-related causes of death, all infections (*10*), and, separately, acquired immunodeficiency syndrome, pneumonia/influenza, and other infectious diseases (Table 1). Occupational diseases, congenital anomalies, conditions related to premature birth, and sudden infant death syndrome were not included because of small numbers.

Analysis of YPLL <65 by Hispanic origin (i.e., Hispanics, non-Hispanic whites, and non-Hispanic blacks) using data from vital records is complicated because of missing information. Based on the CDC criteria for the reporting of deaths by Hispanic origin, YPLL <65 calculations were restricted to the 18 states and the District of Columbia where coverage of Hispanic origin was at least 90% complete (*11*). In 1980, the 18 states and the District of Columbia accounted for 50% of the U.S. white population, 52% of the black population, and 80% of the total Hispanic population (89% of the Mexican population, 78% of the Puerto Rican population, and 34% of the Cuban population). In the calculation of YPLL <65 for American Indians/Alaskan Natives and Asians/Pacific Islanders, as well as for all racial/ethnic groups combined, all states were included.

Death rates per 100,000 U.S. residents (all ages) and YPLL <65 rates per 1,000 U.S. residents <65 years were calculated using data from the 1980 Census and postcensal age-, sex-, and race-specific population estimates for 1987 (*12–16*). Postcensal estimates provide age, sex, and specific Hispanic-origin population figures; they do not provide information by race or state. Data from the 1980 Census provide estimates of the distribution of Hispanics and non-Hispanics by race and state in addition to age,

sex, and specific Hispanic origin. The 1987 population of Hispanics and non-Hispanics by race, age, sex, and specific Hispanic origin was estimated by combining information from 1987 postcensal estimates and 1980 Census figures. Age- and sex-specific estimates of the Hispanic white and black populations were obtained by applying the age- and sex-specific proportions of white Hispanics and black Hispanics in the 1980 Census (*16*) to the 1987 Hispanic population postcensal estimates (*13,14*). Age- and sex-specific estimates of the non-Hispanic white and non-Hispanic black populations were obtained by subtracting age- and sex-specific estimates of numbers of white and black Hispanic persons in 1987 from age- and sex-specific estimates of all white and all black persons in 1987 (*12*). The national population estimates for Hispanics, non-Hispanic whites, and non-Hispanic blacks were extrapolated to the 18 states and the District of Columbia (which meet criteria of mortality reporting by Hispanic origins) on the basis of the proportion of the U.S. total that those states and the District of Columbia represent for blacks, non-Hispanic whites and blacks, Hispanics, and specific Hispanic groups (*11*).

For American Indians/Alaskan Natives and Asians/Pacific Islanders, 1987 sex-specific postcensal estimates (*12*) were used, to which the 1980 Census age and sex distributions of American Indians/Alaskan Natives and Asians/Pacific Islanders were applied (*14*).

RESULTS

Among males, the total YPLL <65 rate per 1,000 population <65 years was highest for non-Hispanic blacks (140.0), followed by American Indians/Alaskan Natives (100.9), Hispanics (74.3), non-Hispanic whites (68.3), and Asians/Pacific Islanders (38.2). Among females, the total YPLL <65 rate was highest for non-Hispanic blacks (73.7),

TABLE 1. Conditions analyzed and corresponding international classification codes* — United States, 1986–1988

Condition	ICD mortality codes
Diseases of the heart	390–398, 402, 404–429
Ischemic heart disease	410–414, 429.2
Stroke	430–434, 436–438
Diabetes	250
Chronic obstructive pulmonary disease	491, 492, 496
All malignant neoplasms	140–208
Lung cancer	162
Breast cancer	174
Cervical cancer	180
Colorectal cancer	153–154
Cirrhosis	571
All infectious diseases	see reference 10
HIV/AIDS†	042–044
Pneumonia/influenza	480–487
Unintentional injury	E800–949
Motor-vehicle related	E810–825
Other	E800–809, E826–949
Homicide	E960–978
Suicide	E950–959
Drug-related conditions	304, 305.2–305.9, 850, E950.0–950.5, E980–980.5

*From *International Classification of Diseases* [ICD], *Ninth Revision*. Washington, DC: Department of Health and Human Services, 1991 (DHSS publication No. [PHS] 91-1260).
†HIV/AIDS = human immunodeficiency virus/acquired immunodeficiency syndrome.

followed by American Indians/Alaskan Natives (52.0), non-Hispanic whites (35.7), Hispanics (32.9), and Asians/Pacific Islanders (23.2) (Figure 1). The rate of total YPLL <65 was 1.9 times greater for all males (74.4) than for all females (39.3); the rate ratio by sex was greatest for Hispanics (2.3 times greater for males than for females) and smallest for Asians/Pacific Islanders (1.6 times greater for males than for females).

The high total YPLL <65 rate for non-Hispanic black males and females reflects high YPLL <65 rates (relative to those in other race and ethnic groups) for most causes of death studied, particularly for homicide (Table 2); homicide accounted for 14% and 6% of total YPLL <65 for non-Hispanic black males and females, respectively. For American Indians/Alaskan Natives, the high YPLL <65 is due to motor-vehicle injuries, other unintentional injuries, suicide, cirrhosis, and diabetes; these five conditions combined accounted for 48% and 34% of the total YPLL <65 for American Indian/Alaskan Native males and females, respectively.

For Hispanic males and females, the rate of YPLL <65 was lower than or similar to rates among non-Hispanic whites for all chronic diseases except cirrhosis (Table 2). For Hispanic males, most of the excess in YPLL <65 (all causes), compared with non-Hispanic white males, was due to homicide, unintentional injuries, cirrhosis, and infections; these four causes accounted for 46% of all YPLL <65 for Hispanic males. The lower rate of YPLL <65 (all causes) for Hispanic females, compared with non-Hispanic white females (Figure 1), is due to lower rates for suicide and unintentional injuries among Hispanic females. The burden of premature mortality varied widely by specific Hispanic origin (Figure 2;Table 3): the rate of YPLL <65 (all causes) was the highest for Puerto Ricans (males, 105.9; females, 39.5), followed by Mexicans (males, 61.4; females, 28.6), and Cubans (males, 61.1; females, 20.4). Among males of all groups, Asians/Pacific Islanders had the lowest rates of YPLL <65; among females, Cubans had the lowest rates.

DISCUSSION

The findings in this surveillance report indicate large differences among YPLL <65 rates by race, Hispanic origin, and sex. They also show that non-Hispanic blacks, who have the highest overall age-adjusted death rates, tend to die at a younger age than members of the following groups (in decreasing order): American Indians/Alaskan Natives, non-Hispanic whites, Hispanics, and Asians/Pacific Islanders.

To calculate years of potential life lost, the 65-year cut-off was used rather than a higher limit because the 65-year cut-off emphasizes premature and preventable deaths (*2*). However, other methods of calculating years of potential life lost have been proposed on the basis of the ages at which social and economic losses are thought to begin and end, as well as on the value of productivity at each age (*17*).

Because of the incomplete reporting of Hispanic origin in some states, analysis of YPLL <65 among Hispanics and non-Hispanic whites and blacks was restricted to 18 states and Washington, D.C.; thus, for those groups, this analysis may not be fully representative of the United States (*11*). The population estimates used to calculate YPLL <65 rates were derived from postcensal estimates and the 1980 Census and may not be completely accurate, particularly for minority groups (*12,19*). In addition, the misclassification of race and ethnicity may also have influenced the results (*19–21*).

Text continued on page 22

FIGURE 1. Rate of years of potential life lost (YPLL) before age 65, by race, Hispanic origin, and sex — United States, 1986–1988

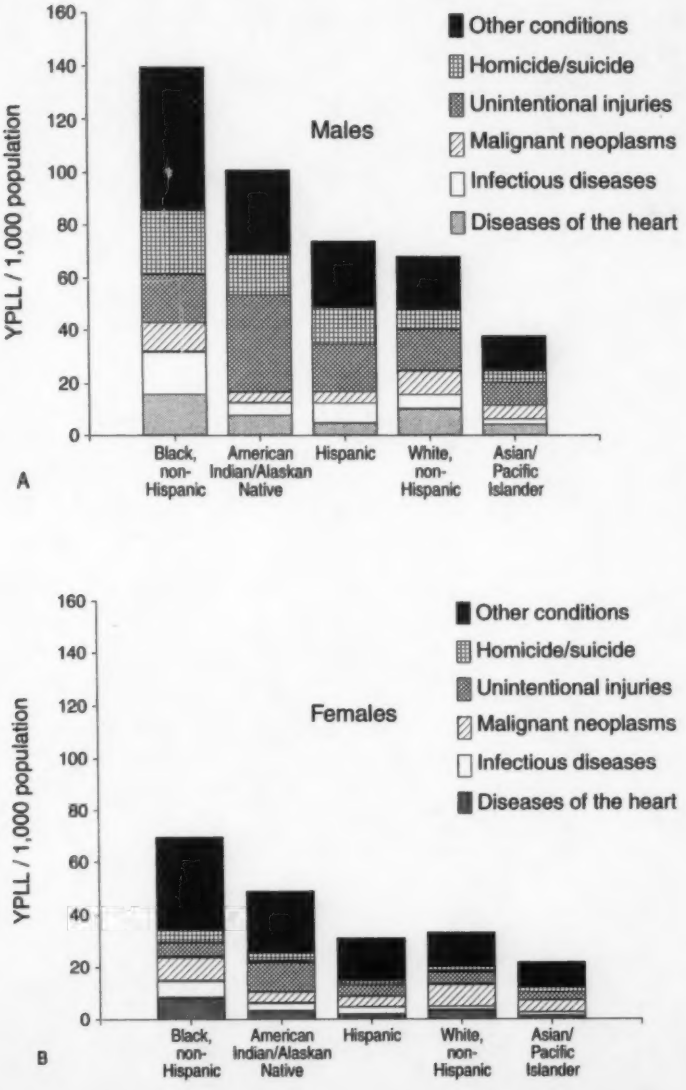


TABLE 2. Age-adjusted death rate/100,000 population (all ages) and YPLL < 65 rate/1,000 persons < 65 years, by race, sex, and Hispanic origin, selected causes of death — United States, 1986–1988

Cause	Total		Non-Hispanic whites*		Non-Hispanic blacks*		Hispanics*		American Indians/Alaskan Natives		Asians/Pacific Islanders	
	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female
All causes												
Death rate	1,104.6	704.8	1,053.8	676.3	1,439.2	890.7	800.3	518.0	969.0	631.2	619.4	398.0
YPLL <65 rate	74.4	39.3	68.3	35.7	140.0	73.7	74.3	32.9	100.9	52.0	38.2	23.2
Ischemic heart disease												
Death rate	310.9	189.1	312.0	188.3	299.2	209.4	190.6	131.7	207.0	117.2	158.7	90.0
YPLL <65 rate	6.9	2.1	7.5	2.1	7.9	3.7	2.8	0.9	4.7	1.4	2.6	0.6
Stroke												
Death rate	62.4	60.3	56.7	55.9	85.1	75.9	44.9	42.3	46.7	43.8	51.4	43.4
YPLL <65 rate	1.2	1.1	1.0	0.9	2.9	2.4	1.0	0.8	1.1	1.0	0.9	0.8
Diabetes												
Death rate	16.1	15.7	14.2	12.9	25.6	31.1	20.8	23.7	28.2	36.0	12.3	10.8
YPLL <65 rate	0.6	0.5	0.6	0.5	1.2	1.1	0.4	0.4	0.9	0.7	0.2	0.2
Chronic obstructive pulmonary disease												
Death rate	47.1	19.5	47.8	21.0	34.1	10.2	20.0	8.8	29.7	11.1†	18.8	5.6
YPLL <65 rate	0.5	0.3	0.5	0.4	0.6	0.3	0.1	0.1	0.2	0.2†	0.1	0.1
Lung cancer												
Death rate	84.0	31.2	80.7	32.1	104.6	30.3	36.3	11.9	44.1	20.9	38.2	16.7
YPLL <65 rate	2.5	1.4	2.6	1.6	3.4	1.4	0.6	0.2	1.0	0.5	0.9	0.5
Breast cancer												
Death rate	—	30.2	—	30.7	—	32.7	—	16.6	—	12.6	12.3	—
YPLL <65 rate	—	2.2	—	2.3	—	2.7	—	1.0	—	0.7	—	1.0
Cervical cancer												
Death rate	—	3.4	—	2.7	—	7.6	—	4.7	—	6.1†	—	3.3
YPLL <65 rate	—	0.4	—	0.4	—	0.7	—	0.4	—	0.6†	—	0.3
Colorectal cancer												
Death rate	27.7	19.8	27.9	19.4	30.4	23.3	13.6	8.9	12.5	11.5	17.5	10.5
YPLL <65 rate	0.7	0.6	0.8	0.6	0.8	0.7	0.3	0.2	0.3†	0.3†	0.4	0.4
Cirrhosis												
Death rate	15.4	6.9	14.5	6.5	25.9	11.1	29.0	9.7	32.9	23.5	6.6	3.9
YPLL <65 rate	1.5	0.7	1.4	0.6	3.4	1.5	2.5	0.6	3.7	2.9	0.5	0.2

Motor-vehicle injury												
Death rate	29.1	11.4	28.0	11.5	27.2	8.2	30.7	8.7	65.8	24.7	17.1	10.5
YPLL <65 rate	10.0	3.7	9.7	3.7	8.5	2.8	10.5	2.9	21.8	8.5	5.3	2.5
Unintentional injuries other than motor-vehicle-related												
Death rate	28.4	11.6	25.5	10.8	42.4	16.3	27.2	8.6	47.5	14.4	12.6	4.5
YPLL <65 rate	6.4	1.9	5.8	1.7	10.1	3.6	7.5	1.8	14.9	4.0	3.4	0.9
Homicide												
Death rate	13.7	4.2	6.5	2.8	58.6	12.9	27.1	4.4	22.8	6.1	6.9	3.1
YPLL <65 rate	4.9	1.5	2.1	1.0	20.9	4.8	10.2	1.6	7.7	2.4	2.3	1.0
Suicide												
Death rate	21.2	5.1	22.5	5.8	11.9	2.4	12.7	2.1	23.5	4.7	9.7	4.3
YPLL <65 rate	5.1	1.3	5.4	1.4	3.4	0.7	3.5	0.6	8.3	1.5	2.4	0.9
Drug-related												
Death rate	3.6	2.2	3.2	2.2	10.2	3.5	6.9	1.6	2.9†	3.0†	0.9†	0.7†
YPLL <65 rate	1.1	0.6	1.0	0.6	2.5	1.0	2.0	0.4	0.8†	0.9†	0.2†	0.2†
All infections												
Death rate	64.4	39.5	62.0	37.7	112.8	56.3	64.9	36.4	67.6	45.5	42.7	26.0
YPLL <65 rate	5.6	2.4	5.3	1.8	17.4	7.3	7.9	2.8	5.0	3.4	2.4	1.4
HIV/AIDS												
Death rate	7.5	0.9	8.1	0.5	25.8	5.0	14.0	1.9	2.1†	0.2†	1.9†	0.2†
YPLL <65 rate	2.2	0.3	2.4	0.2	6.9	1.7	3.7	0.6	0.5†	0.03†	0.5†	0.4†
Pneumonia/influenza												
Death rate	37.3	24.7	36.7	25.3	46.6	24.9	29.8	20.4	41.9	25.0	24.9	15.4
YPLL <65 rate	1.0	0.6	0.9	0.6	3.1	1.8	1.1	0.7	1.6	1.2	0.5	0.3
Infections other than HIV/AIDS and pneumonia/influenza												
Death rate	19.6	13.9	17.2	11.9	40.4	26.4	21.1	14.1	23.6	20.3	15.9	10.4
YPLL <65 rate	2.4	1.5	2.0	1.1	6.2	3.9	2.8	1.7	2.8	2.2	1.4	1.0

* Based on data from 18 states and the District of Columbia (*11*).
† Calculation based on fewer than 100 deaths for the study period (1986–1988).
Abbreviations: YPLL = years of potential life lost; HIV/AIDS = human immunodeficiency virus/acquired immunodeficiency syndrome.

FIGURE 2. Rate of years of potential life lost (YPLL) before age 65, by Hispanic origin and sex — United States, 1986–1988

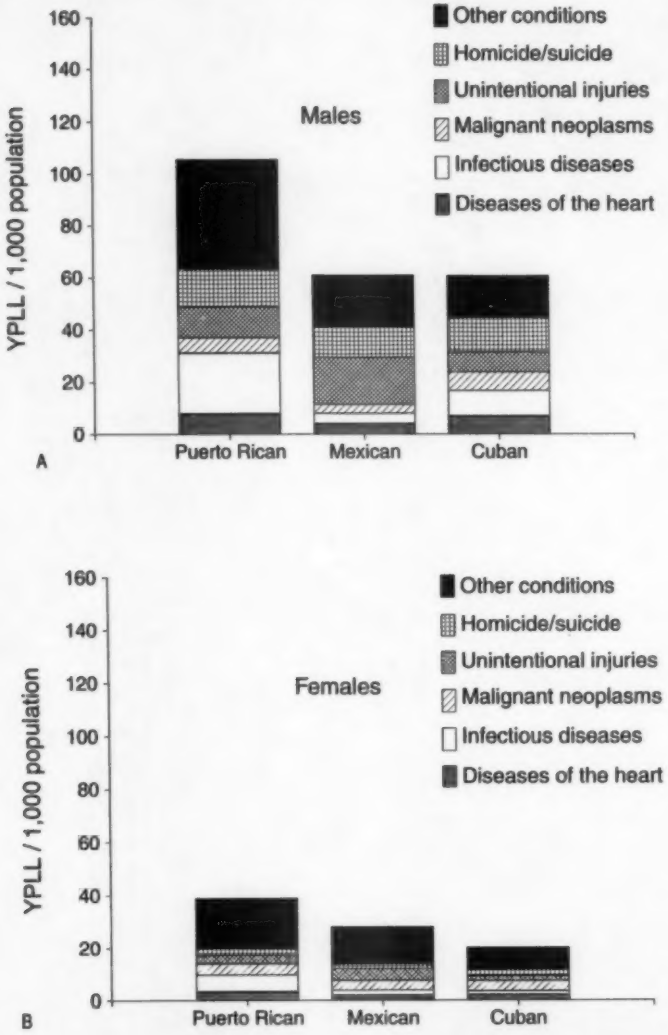


TABLE 3. Age-adjusted death rate/100,000 population (all ages) and YPLL <65 rate/1,000 persons <65 years, by sex and Hispanic origin, selected causes of death — United States, 1986–1988*

Cause	Mexican		Puerto Rican		Cuban	
	Male	Female	Male	Female	Male	Female
All causes						
Death rate	448.2	319.1	1165.0	703.3	644.9	323.8
YPLL <65 rate	61.4	28.6	105.9	39.5	61.1	20.4
Ischemic heart disease						
Death rate	109.9	85.1	290.7	217.0	169.5	92.0
YPLL <65 rate	2.1	0.7	4.7	1.6	4.8	1.1
Stroke						
Death rate	21.7	22.6	49.2	41.3	30.2	24.2
YPLL <65 rate	0.8	0.7	1.5	1.0	1.5	0.8
Diabetes						
Death rate	10.0	12.3	31.6	32.7	9.8†	11.1†
YPLL <65 rate	0.4	0.4	0.8	0.6	0.6†	0.3†
Chronic obstructive pulmonary disease						
Death rate	12.0	6.4	32.8	15.5	15.2†	5.4†
YPLL <65 rate	0.1	0.0	0.3	0.2	0.1†	0.1†
Lung cancer						
Death rate	20.1	7.1	42.3	15.8	40.6	6.8†
YPLL <65 rate	0.4	0.2	0.9	0.3	1.7	0.3†
Breast cancer						
Death rate	—	10.8	—	18.5	—	15.3
YPLL <65 rate	—	0.9	—	1.0	—	1.3
Cervical cancer						
Death rate	—	2.8	—	5.4†	—	2.2†
YPLL <65 rate	—	0.4	—	0.4†	—	0.3†
Colorectal cancer						
Death rate	8.3	6.7	18.9	13.8	13.7†	10.6†
YPLL <65 rate	0.2	0.2	0.5	0.3	0.5†	0.3†
Cirrhosis						
Death rate	17.7	5.7	50.6	12.0	10.6†	4.5†
YPLL <65 rate	1.8	0.5	5.4	1.0	0.9	0.3
Motor-vehicle injury						
Death rate	11.9	4.84	17.8	5.4	14.5†	3.6†
YPLL <65 rate	11.0	3.0	4.9	1.5	4.6†	0.9†
Unintentional injuries other than motor-vehicle-related						
Death rate	14.1	5.9	36.1	10.9	14.7†	6.1†
YPLL <65 rate	7.1	1.7	7.4	1.8	3.2†	1.1†
Homicide						
Death rate	15.9	3.4	35.0	6.5	32.6	6.2†
YPLL <65 rate	8.6	1.2	11.7	2.4	10.5	2.0†
Suicide						
Death rate	7.0	1.3	11.7	1.2†	14.9†	2.3†
YPLL <65 rate	3.2	0.6	2.8	0.3†	3.0†	0.5†
Drug-related						
Death rate	6.0	1.5	23.5	3.6	2.2†	1.7†
YPLL <65 rate	1.2	0.3	6.3	1.2	0.7†	0.3†
All infections						
Death rate	44.6	25.4	146.6	64.2	60.6	19.5
YPLL <65 rate	4.0	2.0	23.3	6.6	9.8	1.4
HIV/AIDS						
Death rate	15.7	2.7	57.2	10.0	24.2	1.2†
YPLL <65 rate	1.2	0.1	15.1	3.3	6.3	0.2†
Pneumonia/influenza						
Death rate	16.0	13.8	49.0	32.9	19.5†	11.8†
YPLL <65 rate	0.7	0.5	2.8	1.4	0.7†	0.4†
Infections other than HIV/AIDS and pneumonia/influenza						
Death rate	12.9	8.9	40.4	30.3	16.9	6.5
YPLL <65 rate	2.1	1.4	5.4	1.9	2.8	0.8

* Based on data from 18 states and the District of Columbia (*11*).
† Calculation based on fewer than 100 deaths for the study period (1986–1988).

Abbreviations: YPLL = years of potential life lost; HIV/AIDS = human immunodeficiency virus/acquired immunodeficiency syndrome.

Therefore, the rates reported should be interpreted with caution, particularly for groups other than non-Hispanic whites and blacks.

Elevated YPLL <65 rates may reflect a higher incidence of disease in younger age groups or a higher case-fatality rate at young ages. The former may indicate a greater prevalence of preventable risk factors early in life; the latter may indicate more advanced disease at diagnosis, co-morbidity, delay in treatment, or other gaps in the quality of health care received. For causes of death such as ischemic heart disease, lung cancer, cirrhosis, and chronic obstructive pulmonary disease, the differences among YPLL <65 rates reported among racial and ethnic groups may reflect wide differences in incidence among young adults that are influenced by age, race, and ethnic differences in behaviors such as smoking, alcohol use, diet, and physical exercise (*5,18*). However, differences in incidence at younger ages fail to fully explain the dramatic variation of YPLL <65 within racial and ethnic groups. For instance, although incidence of breast cancer is lower for black than for white females, the age-adjusted death rate is higher for blacks because of a lower survival rate, even if the stage of disease at diagnosis is the same (*5,22*). In addition, our findings suggest that black females tend to die of breast cancer at younger ages than white females. Therefore, it seems likely that breast cancer screening and adequate treatment are less accessible to or less utilized by blacks — particularly those in younger age groups. This gap may also apply to conditions such as cervical and colorectal cancer (*22*). The low age-adjusted death rates and YPLL <65 rates for most conditions studied among Cubans and Asians/Pacific Islanders of both sexes suggest that these populations have a greater survival rate or that behaviors that may adversely affect health are less common than in other groups.

The wide differential of YPLL <65 rates by race and Hispanic origin for specific causes of death for which effective primary and secondary preventive measures are available (e.g., ischemic heart disease; stroke; lung, breast, cervical, and colorectal cancer; pneumonia/influenza) is consistent with other reports of unmet health-prevention and health-care needs for black Americans, American Indians/Alaskan Natives, and Hispanics (*23*). Because the causes of death in this report are preventable or can be delayed (*9*), substantial improvement in reducing premature mortality can be achieved by targeted and intensified intervention. Because the high rates of premature death in different groups are due to different conditions, it is essential that these specific causes be considered in determining health-care priorities and designing prevention strategies.

References

1. CDC. Introduction to table V: premature deaths, monthly mortality, and monthly physician contacts — United States. MMWR 1982;31:109–10.
2. CDC. Premature mortality in the United States: public health issues in the use of years of potential life lost. MMWR 1986;35(No. 2S).
3. Romeder JM, McWhinnie JR. Potential years of life lost between ages 1 and 70: an indicator of premature mortality for health planning. Int J Epidemiol 1977;6:143–51.
4. Perloff JD, LeBailly SA, Kletke PR, Budetti PP, Connelly JP. Premature death in the United States: years of life lost and health priorities. J Public Health Policy 1984;5:167–84.
5. National Center for Health Statistics. Health, United States, 1990. Hyattsville, MD: Public Health Service, 1991.
6. National Center for Health Statistics. Mortality detail, 1986 [machine-readable public-use data tape]. Hyattsville, MD: Department of Health and Human Services, 1988.

7. National Center for Health Statistics. Mortality detail, 1987 [machine-readable public-use data tape]. Hyattsville, MD: Department of Health and Human Services, 1989.
8. National Center for Health Statistics. Mortality detail, 1988 [machine-readable public-use data tape]. Hyattsville, MD: Department of Health and Human Services, 1990.
9. Hahn RA, Teutsch SM, Rothenberg RB. Chronic disease reports from the MMWR, vol. 38 (1989) and vol. 39 (1990) [compilation]. Atlanta, GA: Public Health Service, Centers for Disease Control, 1990.
10. Jason JM, Jarvis WR. Infectious diseases: preventable causes of infant mortality. Pediatrics 1987;80:335–41.
11. National Center for Health Statistics. Vital statistics of the United States, 1987, vol. II — mortality: technical appendix. Hyattsville, MD: Department of Health and Human Services, 1989:6.
12. Bureau of the Census. United States population estimates by age, sex, and race: 1980 to 1987. Washington, DC: Department of Commerce, 1988 (publication P-25, No. 1022).
13. Bureau of the Census. United States population estimates by age, sex, race and Hispanic origin: 1980 to 1988. Washington, DC: Department of Commerce, 1990 (publication P-25, No. 1045).
14. Bureau of the Census. General population characteristics, United States summary, 1980 census of population. Washington, DC: Department of Commerce, 1981 (publication PC-80-1-B1).
15. Bureau of the Census. The Hispanic population in the United States: March 1986 and 1987. Washington, DC: Department of Commerce, 1988 (publication P-20, No. 434).
16. Bureau of the Census. Census of population and housing, 1980: county population by age, sex, race, and Spanish origin. Washington, DC: Department of Commerce, 1983 (unpublished report).
17. Gardner JW, Sanborn JS. Years of potential life lost (YPLL) — what does it measure? Epidemiology 1990;1:322–9.
18. US Department of Health and Human Services: Report of the Secretary's Task Force on Black & Minority Health. Washington, DC: Department of Health and Human Services, 1985.
19. Hahn R. The state of federal health statistics on racial and ethnic groups. JAMA 1992;267:268–71.
20. Hahn RA, Mulinare J, Teutsch SM. Inconsistencies in coding of race and ethnicity between birth and death in US infants: a new look at infant mortality, 1983 through 1985. JAMA 1992;267:259–63.
21. Kleinman JC. Infant mortality among racial/ethnic minority groups, 1983–1984. MMWR 1990;39(No. SS-2):31–9.
22. National Cancer Institute. Cancer among blacks and other minorities: statistical profiles. Bethesda, MD: Public Health Service, 1986 (National Institutes of Health publication No. 86-2785).
23. Schwartz E, Kofie VY, Rivo M, Tuckson RV. Black/white comparisons of deaths preventable by medical intervention: United States and the District of Columbia 1980–1986. Int J Epidemiol 1990;19:591–8.

Group B Streptococcal Disease in the United States, 1990: Report from a Multistate Active Surveillance System

Kenneth M. Zangwill, M.D.
Anne Schuchat, M.D.
Jay D. Wenger, M.D.
Meningitis and Special Pathogens Branch
Division of Bacterial and Mycotic Diseases
National Center for Infectious Diseases
and the
*Group B Streptococcal Disease Study Group**

Summary

Group B streptococcal (GBS) disease is the most common cause of neonatal sepsis and meningitis in the United States. It is also an important cause of morbidity among pregnant women and adults with underlying medical conditions. Because most states have not designated GBS disease as a reportable condition, previous estimates of the incidence of GBS disease were based on studies from single hospitals or small geographic areas. This report summarizes the results of population-based active surveillance for invasive GBS disease in counties within four states that had an aggregate population of 10.1 million persons in 1990. A case of GBS disease was defined as isolation of group B streptococcus from a normally sterile anatomic site in a resident of one of the surveillance areas.

Age- and race-adjusted projections to the U.S. population suggest that >15,000 cases and >1,300 deaths due to GBS disease occur each year. The projected age- and race-adjusted national incidence is 1.8/1,000 live births for neonatal GBS disease and 4.0/100,000 population per year for adult GBS disease. Intrapartum chemoprophylaxis for pregnant women at risk for delivering infants with GBS disease is the most effective strategy available for prevention of neonatal disease. Development of effective GBS vaccines may prevent GBS disease in both infants and adults. Ongoing surveillance for GBS disease is important for targeting preventive measures and determining their effectiveness.

INTRODUCTION

Group B streptococcal (GBS) disease is an important cause of morbidity and mortality in newborns and in adults with underlying disease such as malignancy and diabetes mellitus. GBS disease also causes substantial illness in pregnant women,

---

*Members of the Group B Streptococcal Disease Study Group include Katherine A. Deaver and Richard Pierce, M.P.H., CDC; Gretchen Anderson, M.P.H., Kevin Kraus, Elizabeth Stone, M.P.H., and Arthur Reingold, M.D., San Francisco (California) Department of Health; Margaret Rados, Louis Lefkowitz, M.D., Department of Preventive Medicine, Vanderbilt Medical Center, Nashville, Tennessee; Pam Archer, M.P.H., Jane Strack, and Gregory Istre, M.D., Oklahoma State Department of Health, Oklahoma City, Oklahoma; Christopher Harvey, M.P.H., Tina Stull, M.D., Monica Farley, M.D., and David Stephens, M.D., Emory University Department of Medicine, Atlanta, Georgia.

including chorioamnionitis, stillbirth, and wound infections. In recent years, development and evaluation of prevention strategies for GBS disease have increased. Information on the magnitude of GBS disease in the general population, however, has not been available. This report summarizes information from a population-based active surveillance system for invasive GBS disease conducted in a large U.S. population from January 1 through December 31, 1990.

METHODS

Active surveillance was conducted in an aggregate population of 10.1 million persons consisting of residents of the San Francisco area (three counties), four metropolitan counties in Tennessee, eight counties in the metropolitan area of Atlanta, and the entire state of Oklahoma. Racial distribution of the surveillance population was 71% white, 18% black, and 11% other. Hispanic persons accounted for 3% of the population.

A case was defined as the isolation of group B streptococci from a normally sterile site (e.g., blood, cerebrospinal fluid [CSF]) from a resident of the areas under surveillance. More than 99% (264/265) of the hospitals in the surveillance areas participated. On a biweekly basis, surveillance workers requested standardized reports of cases of invasive GBS disease from contacts in each hospital laboratory in the surveillance areas. Laboratory records in all hospitals in the Atlanta surveillance area were audited in 1990 to estimate completeness of reporting and to detect additional cases. Limited audits were performed in the other surveillance areas.

Total incidence and age- and race-specific rates were calculated for each surveillance area using 1990 population data from the U.S. Bureau of the Census. The National Center for Health Statistics' provisional data for 1990 was the source of live birth information used in national projections. National estimates of GBS disease were calculated by multiplying the age- and race-specific incidence in the combined surveillance areas by the appropriate U.S. population figures for these categories.

RESULTS

Total Incidence of GBS Disease

In 1990, 635 case-patients with invasive GBS disease were identified (Table 1). The incidence was highest among infants <90 days of age (Figure 1). Among case-patients for whom data were available, 323/596 (54.2%) were white, 355/633 (56.1%) were female, and 34/508 (6.7%) were Hispanic. The incidence of disease was significantly

TABLE 1. Group B streptococcal disease, by race — selected U.S. counties,* 1990

Race†	Cases		Deaths	
	Number	Rate§	Number	Case-fatality (%)
White	323	4.5	28	9.2
Black	251	13.5	25	11.8
All races	635	6.4	60	10.5

*Surveillance areas included counties in California, Georgia, and Tennessee and the entire state of Oklahoma.
† $p<0.0001$, blacks compared with whites.
§ Cases per 100,000 population.

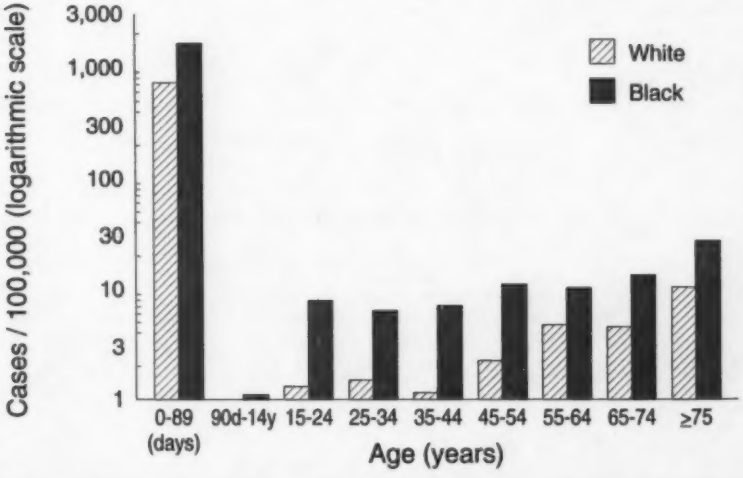
higher among blacks than whites in all age groups ($p < 0.0001$). The crude incidence of GBS disease was higher among white Hispanics than among the non-Hispanic white population (10.5/100,000 [95% confidence interval (CI) = 7.0,15.1] versus 3.5/100,000 [CI = 3.1,3.9]). Of 574 cases for which outcome information was available, 60 (10.5%) resulted in death. There was no apparent seasonal clustering of GBS disease.

Infant Disease

Of the 635 total cases, 306 (48.2%) occurred among infants <90 days of age, an incidence of 1.7/1,000 live births (Table 2). GBS disease in infants is characterized as either early-onset (occurring in infants <7 days old) or late-onset (occurring in infants 7–89 days old). Two hundred forty-seven (80.7%) infants with early-onset disease and 59 (19.3%) with late-onset disease were identified. Three infants had separate episodes of both early-onset and late-onset disease. One hundred fifty-two of 306 infants (53.7%) were white and 123 (43.5%) were black. For five infants, GBS disease occurred between 90 days and 1 year of life.

The case-fatality rate for infant disease was 5.8% (16/278) (Table 2). The rate was similar among those with early-onset disease (5.7%) and late-onset disease (6.0%). Thirteen (81.3%) deaths occurred among babies born <34 weeks of gestation, and eight of these infants were black. Although black infants were significantly more likely to die than were white infants (relative risk [RR] = 4.1, CI = 1.2,14.6), prematurity may have accounted for this finding.

FIGURE 1. Incidence of group B streptococcal disease, by race and age, selected U.S. counties,* 1990



*Active surveillance areas included counties in California, Georgia, and Tennessee and the entire state of Oklahoma.

Information on gestational age was available for only 152 (50%) infants. Twenty (13%) were born at <34 weeks of gestation, 23 (15%) at 34–36 weeks, and 109 (72%) at ≥37 weeks (Table 2). For comparison, in 1989, 10.3% of the births in the United States occurred at <37 weeks of gestational age. For infants with GBS disease for whom information was available, prematurity (gestational age <37 weeks) was more common among blacks (24/56) than whites (14/78; p<0.003).

Of the infants identified with GBS disease, 294 (96%) had bacteremia and 33 (11%) had meningitis. Among the 247 infants with early-onset disease, group B streptococci were isolated from blood samples from 241 (98%) patients and from CSF from 14 (4.6%) patients (Table 3). Of the 59 infants with late-onset disease, bacteremia was present in 53 (90%) and CSF was positive for group B streptococci in 19 (32%; Table 3).

TABLE 2. Group B streptococcal disease among infants <90 days old — selected U.S. counties,* 1990

	Early-onset disease†		Late-onset disease		Total disease	
Incidence§¶	**No.**	**(rate)**	**No.**	**(rate)**	**No.**	**(rate)**
White	129	(1.1)	23	(0.19)	152	(1.3)
Black	92	(2.0)	31	(0.67)	123	(2.7)
All races	247	(1.4)	59	(0.32)	306	(1.7)
Deaths	**No.**	**(%)**	**No.**	**(%)**	**No.**	**(%)**
White	3	(2.4)	0		3	(2.1)
Black	7	(8.6)	3	(13)	10	(9.0)
All races	13	(5.7)	3	(6.0)	16	(5.8)
Gestational age**	**No.**	**(%)**	**No.**	**(%)**	**No.**	**(%)**
<34 weeks	16	(12)	4	(22)	20	(13)
34–36 weeks	19	(14)	4	(22)	23	(15)
≥37 weeks	99	(74)	10	(56)	109	(72)

*Surveillance areas included counties in California, Georgia, and Tennessee and the entire state of Oklahoma.
†Early and late onset defined as occurrence <7 days and 7–89 days of life, respectively.
§Cases per 1,000 live births.
¶p<0.0001, blacks compared with whites for early and late onset disease.
**Data available for only 152 (50%) of cases.

TABLE 3. Clinical site of isolation, group B streptococcal disease — selected U.S. counties,* 1990

	Infants, early-onset disease (N=247)†		Infants, late-onset disease (N=59)		Nonpregnant adults§ (N=227)		Pregnant adults (N=67)		Total (N=600)	
Site of isolation	**No.**	**(%)**	**No.**	**(%)**	**No.**	**(%)**	**No.**	**(%)**	**No.**	**(%)**
Blood	241	(98)	53	(90)	205	(90)	53	(79)	552	(92)
Cerebrospinal fluid	14	(4.6)	19	(32)	6	(2.6)	0		39	(6.5)
Amniotic fluid/placenta	1	(0.4)	0				19	(28)	20	(3.3)
Other	7	(2.8)	0		28	(12)	2	(3.0)	37	(6.2)

*Surveillance areas included counties in California, Georgia, and Tennessee and the entire state of Oklahoma.
†Early and late onset defined as occurrence <7 days and 7–89 days of life, respectively.
§≥15 years of age.

Adult Disease

The incidence of GBS disease among adults (defined as persons ≥15 years of age) was 3.6/100,000 population (Table 4). Of 307 adult case-patients with GBS disease, 67 (11%) were pregnant women. One hundred sixty of 307 adult case-patients (52%) were white and 123 (40%) were black. The incidence of GBS disease increased with age (Figure 1).

Among case-patients for whom data were available, there were 265/307 (86.3%) adult case-patients with bacteremia and 9/279 (3.2%) with a positive CSF culture (Table 3). (Those women with unknown pregnancy status [seven with bacteremia and three with meningitis] are not listed in Table 3.) Of 227 cases in nonpregnant adults, 205 (90.3%) were associated with bacteremia and 6/205 (2.6%) had a positive CSF culture (Table 3). Other clinical diagnoses in nonpregnant adults included cellulitis in 19 (8.4%), pneumonia in 13 (5.7%), peritonitis in 10 (4.4%), and arthritis in five (2.2%). Among pregnant women, group B streptococcus was isolated from blood samples from 53/67 (79.1%) patients and from amniotic fluid or placenta from 19 (28.4%) patients. No pregnant women had meningitis, and chorioamnionitis was the most frequent clinical diagnosis (32.8%).

Outcome of the pregnancy was known for 43 women. Twenty-one (48.8%) women delivered infants with no apparent illness, 15 (34.9%) delivered infants with nonfatal neonatal GBS disease, four (9.3%) delivered infants who died as newborns from GBS disease, and three (7.0%) pregnancies resulted in stillbirth. Information on the use of intrapartum antibiotics was not available.

The case-fatality rate for disease among adults was 43/275 (16%). Adults were more likely to die as a result of GBS infection than were infants (RR = 2.7, CI = 1.6,4.7). Forty-two of 242 (17.4%) adult case-patients with bacteremia, 4/7 (57.1%) with meningitis, and 4/12 (33.3%) with pneumonia died. Death was more likely to occur among adults who had meningitis compared with those who did not (RR = 3.9, CI = 2.0,8.0) or among adults ≥65 years of age compared with adults <65 (RR = 3.9, CI = 2.2,7.1). No pregnant case-patients died.

Of the 221 cases detected in the Atlanta area by the audit, 122 (55%) were reported through the surveillance system. Limited audits performed on randomly selected hospitals in the other surveillance areas suggested that the surveillance system detected 65%–80% of case-patients. The case-patients detected at audit were included in the number of cases reported here and did not differ from other case-patients with respect to distribution by race, age, or sex.

TABLE 4. Group B streptococcal disease among adults ≥15 years, by race — selected U.S. counties,* 1990

	Cases§		Deaths	
Race†	No.	Rate	No.	Case-fatality (%)¶
White	160	2.7	24	16
Black	123	9.0	15	15
All races	307	3.6	43	16

*Surveillance areas included counties in California, Georgia, and Tennessee and the entire state of Oklahoma.
† p<0.0001, blacks compared with whites.
§ Cases per 100,000 population per year.
¶ Percentage of the 275 cases with known outcome.

Based on data from the surveillance system, age- and race-adjusted projections for the entire U.S. population suggest that, in 1990, >15,000 cases of GBS disease occurred in the United States (Table 5). Of these, 7,600 (50%) cases and 310 deaths occurred among infants ≤90 days of age, and 7,600 (50%) cases and 940 deaths occurred among adults.

DISCUSSION

Because GBS disease is not a reportable condition in most states, estimates of its incidence were previously based on studies performed in small geographic areas or single hospital populations not necessarily representative of the heterogeneous U.S. population. In contrast, the population-based active surveillance system described in this report was designed to detect all cases of GBS disease diagnosed in a specific geographic area. Rates of GBS disease were lower in other surveillance areas than in Atlanta, where prospective surveillance was supplemented by a complete laboratory audit. The audit suggested that active surveillance in Atlanta detected approximately 55% of all cases. In the other surveillance areas, only limited audits were performed; therefore, the projections in this report represent a minimum estimate of the true burden of GBS disease. These findings illustrate the difficulty of obtaining accurate incidence data on GBS disease. Complete audits in all surveillance areas for 1991 are currently under way to provide a more complete estimate of the burden of GBS disease.

The age- and race-adjusted incidences projected for the United States are lower than those in most previous reports (*1*). This apparent decrease probably does not reflect a failure to detect cases, however, since the incidence in urban centers was similar to that reported previously, suggesting that the overall lower incidence may reflect a lower burden of disease outside inner-city areas. Overall lower incidences can likely be accounted for by the racial and geographic diversity of the population under surveillance and the wide variety of hospitals included in surveillance.

Bacteremia is the most frequent presentation in both infants and adults. Meningitis occurs more commonly in late-onset than in early-onset disease, but it accounts for only about 10% of infections in infants and <5% of disease in adults. The relative frequency of clinical presentations in adult disease is similar to that of other series (*2–4*). Pregnant women are at particular risk of morbidity from GBS disease; however, this

TABLE 5. Projections of incidence of and mortality from group B streptococcal disease — United States, 1990*

	Infants, early-onset disease†		Infants, late-onset disease†		Total disease among infants <90 days		Adults ≥15 years		Total disease among all ages	
All races (incidence)§	6,200	(1.5)	1,400	(0.35)	7,600	(1.8)	7,600	(4.0)	15,000	(6.2)
Deaths (%)	260	(4.3)	47	(3.2)	310	(4.1)	940	(12.4)	1,300	(8.2)

*Age- and race-adjusted projections.
†Early and late onset defined as occurrence <7 days and 7–89 days of life, respectively.
§Incidence per 1,000 live births for infant disease and per 100,000 population for adult and total disease.

report focuses on invasive GBS disease, which does not represent the total number of GBS infections in pregnant women.

Rates of disease were higher among blacks than among whites for all age groups. Blacks have previously been shown to have higher rates of neonatal infection than whites independent of the effects of low birth weight, prematurity, and young maternal age (*5*). A study of adult disease in metropolitan Atlanta reported an increased risk of GBS disease in blacks compared with whites (*2*). In this surveillance system, the potential effects of other factors — such as socioeconomic status or birth weight (*6*) — on rates of disease could not be evaluated. This report demonstrates that most infants with GBS disease are not premature, but data on gestational age were available for only half of the infant case-patients. Further studies are needed to determine reasons for the racial differences in the incidence of GBS disease.

Age- and race-adjusted case-fatality projections for neonatal disease were lower than those reported previously (*1,7*), particularly in comparison with the 15%–50% case-fatality rate observed in studies from the 1970s (*8–10*). Recent improvement in neonatal care is probably the most important factor contributing to reduced mortality rates among infants (*11*).

Randomized clinical trials have demonstrated that administration of intrapartum chemoprophylaxis to pregnant women colonized with group B streptococci who experience fever, preterm labor, or prolonged rupture of membranes reduces the likelihood of neonatal early-onset infection as well as postpartum maternal morbidity (*12–14*). This preventive strategy requires detection of GBS infection in pregnant women. Fifteen percent to 40% of all pregnant women are colonized with group B streptococci in the vagina or rectal area (*1*), which may result in intrauterine transmission to the fetus (*10*) and subsequent neonatal infection or fetal loss. Approximately 90%–95% of women colonized with group B streptococci at delivery can be detected as carriers through vaginal and rectal cultures obtained during the second trimester (usually 26–28 weeks) (*13*). Rapid detection of GBS antigen from vaginal specimens collected at the time of delivery may permit detection of GBS colonization when prenatal screening is not available (*15*). Administration of intrapartum antibiotics to all pregnant women with GBS infection would likely lead to unnecessary costs and adverse reactions; in contrast, the selective use of intrapartum antibiotics for colonized women who experience perinatal complications has been shown to be cost-effective (*16,17*).

Although chemoprophylaxis is effective in preventing early-onset disease in neonates, even the most successful chemoprophylaxis regimens do not prevent all disease due to group B streptococci. Hope for further impact on neonatal disease, as well as prevention of adult disease, lies in development of GBS vaccines (*18*). Several vaccines designed to induce antibody response against the polysaccharide capsule of group B streptococcus are currently being developed.

The American College of Obstetricians and Gynecologists has reviewed the utility of chemoprophylactic regimens for the prevention of GBS disease and, in light of the magnitude of the disease and the economic burden in the United States, considers such regimens justifiable (*19*). Also, the American Academy of Pediatrics has formulated recommendations regarding proposed strategies for prevention of neonatal and postpartum disease (*20*). Continued and improved surveillance is critical for identifying groups at increased risk, targeting preventive measures, demonstrating the efficacy of these measures, and confirming their impact.

References

1. Baker CJ, Edwards MS. Group B streptococcal infections. In: Remington JS, Klein JO, eds. Infectious diseases of the fetus and newborn infant. 3rd edition. Philadelphia: WB Saunders 1990:742–811.
2. Schwartz B, Schuchat A, Oxtoby MJ, Cochi SL, Hightower A, Broome CV. Invasive group B streptococcal disease in adults. JAMA 1991;266:1112–4.
3. Opal SM, Cross A, Palmer M, Almazan R. Group B streptococcal sepsis in adults and infants: contrasts and comparisons. Arch Intern Med 1988;148:641–5.
4. Dworzack DL, Hodges GR, Barnes WG, Rosett W. Group B streptococcal infections in adult males. Am J Sci 1979;277:67–73.
5. Schuchat A, Oxtoby M, Cochi SL, Sikes RK, Hightower A, Plikaytis B, Broome CV. Population-based risk factors for neonatal group B streptococcal disease: results of a cohort study in metropolitan Atlanta. J Infect Dis 1990;162:672–7.
6. Cochi SL, Feldman RA. Estimating national incidence of group B streptococcal disease: the effect of adjusting for birth weight. Pediatr Infect Dis J 1983;2:414–5.
7. Dillon HC Jr, Khare S, Gray BM. Group B streptococcal carriage and disease: a 6-year prospective study. J Pediatr 1987;110:31–6.
8. Pass MA, Gray BM, Khare S, Dillon HC Jr. Prospective studies of group B streptococcal infections in infants. J Pediatr 1979;95:437–43.
9. Franciosi RA, Knostman JD, Zimmerman RA. Group B streptococcal neonatal and infant infections. J Pediatr 1973;82:707–18.
10. Baker CJ, Barrett FF. Transmission of group B streptococci among parturient women and their neonates. J Pediatr 1973;83:919–25.
11. Lannering B, Larsson LE, Rojas J, Stahlman MT. Early onset group B streptococcal disease. Seven year experience and clinical scoring system. Acta Paediatr Scand 1983;72:597–602.
12. Boyer KM, Gotoff SP. Prevention of early-onset neonatal group B streptococcal disease with selective intrapartum chemoprophylaxis. N Engl J Med 1986;314:1665–9.
13. Boyer KM, Gotoff SP. Antimicrobial prophylaxis of neonatal group B streptococcal sepsis. Clin Perinatol 1988;15:831–50.
14. Morales WJ, Lim D. Reduction of group B streptococcal maternal and neonatal infections in preterm pregnancies with premature rupture of membranes through a rapid identification test. Am J Obstet Gynecol 1987;157:13–6.
15. Wald ER, Dashefsky B, Green M, Harger J, Parise M, Korey C, Byers C. Rapid detection of group B streptococci directly from vaginal swabs. J Clin Microbiol 1987;25:573–4.
16. Strickland DM, Yeomans ER, Hankins GDV. Cost-effectiveness of intrapartum screening and treatment for maternal group B streptococci colonization. Am J Obstet Gynecol 1990;163:4–8.
17. Mohle-Boetani JC, Schuchat A, Plikaytis BD, Smith D, Broome CV. Comparison of preventive strategies for neonatal group B streptococcal (GBS) infection: a cost-effectiveness analysis [abstract 1058]. Interscience Conference on Antimicrobial Agents and Chemotherapy, Chicago, September 29–October 2, 1991.
18. Baker CJ, Rench MA, Edwards MS, Carpenter RJ, Hays BM, Kasper DL. Immunization of pregnant women with a polysaccharide vaccine of group B streptococcus. N Engl J Med 1988;319:1180–5.
19. American College of Obstetricians and Gynecologists. Group B streptococcal infections in pregnancy. Washington, DC: ACOG Technical Bulletin [No. 170], July 1992.
20. American Academy of Pediatrics. Guidelines for prevention of group B streptococcal infection by chemoprophylaxis. Pediatrics 1992; 90:775–8.

State and Territorial Epidemiologists and Laboratory Directors

State and Territorial Epidemiologists and Laboratory Directors are gratefully acknowledged for their contributions to this report. The epidemiologists listed below were in the positions shown as of September 4, 1992, and the laboratory directors listed below were in the positions shown as of August 1992.

State/Territory	Epidemiologist	Laboratory Director
Alabama	Charles H. Woernle, MD, MPH	William J. Callan, PhD
Alaska	John P. Middaugh, MD	Katherine A. Kelley, DrPH
Arizona	Larry Sands, DO, MPH (Acting)	Barbara J. Erickson, PhD
Arkansas	Thomas C. McChesney, DVM	Robert L. Horn
California	George W. Rutherford, MD	Michael G. Volz, PhD
Colorado	Richard E. Hoffman, MD, MPH	Ronald L. Cada, DrPH
Connecticut	James L. Hadler, MD, MPH	Sanders F. Hawkins, PhD (Acting)
Delaware	A. LeRoy Hathcock, Jr., Ph.D.	Mahadeo P. Verma, PhD
District of Columbia	Martin E. Levy, MD, MPH	James B. Thomas, ScD
Florida	Richard S. Hopkins, MD, MSPH	E. Charles Hartwig, ScD
Georgia	Joseph A. Wilber, MD	Elizabeth A. Franko, PhD
Hawaii	Richard L. Vogt, MD	Vernon K. Miyamoto, PhD
Idaho	Fritz R. Dixon, MD	Richard H. Hudson, PhD
Illinois	Byron J. Francis, MD, MPH	David F. Carpenter, PhD
Indiana	Mary Lou Fleissner, DrPH	Gregory V. Hayes, DrPH
Iowa	Laverne A. Wintermeyer, MD	W. J. Hausler, Jr, PhD
Kansas	Andrew R. Pelletier, MD	Roger H. Carlson, PhD
Kentucky	Reginald Finger, MD, MPH	Thomas E. Maxson, DrPH
Louisiana	Louise McFarland, DrPH	Henry B. Bradford, Jr, PhD
Maine	Kathleen F. Gensheimer, MD	Philip W. Haines, DrPH
Maryland	Ebenezer Israel, MD, MPH	J. Mehsen Joseph, PhD
Massachusetts	Alfred DeMaria, Jr, MD	Ralph J. Timperi, MPH
Michigan	Kenneth R. Wilcox, Jr, MD, DrPH	Robert Martin, DrPH
Minnesota	Michael T. Osterholm, PhD, MPH	Pauline Bouchard, JD, MPH
Mississippi	F. E. Thompson, Jr, MD, MPH	R. H. Andrews, MPH
Missouri	H. Denny Donnell, Jr, MD, MPH	Eric C. Blank, DrPH
Montana	Todd Damrow, PhD, MPH	Douglas Abbott, PhD
Nebraska	Thomas J. Safranek, MD	John Blosser
Nevada	Debra Brus, DVM	Arthur F. DiSalvo, MD
New Hampshire	M. Geoffrey Smith, MD, MPH	Veronica C. Malmberg
New Jersey	Kenneth C. Spitalny, MD	Shahiedy I. Shahied, PhD
New Mexico	C. Mack Sewell, DrPH, MS	Loris W. Hughes, PhD
New York City	Kenneth R. Ong, MD	Stanley Reimer
New York State	Guthrie Birkhead, MD	Lawrence Sturman, MD, PhD
North Carolina	J. Newton MacCormack, MD, MPH	Samuel N. Merritt, DrPH
North Dakota	Larry Shireley, MS, MPH	James L. Pearson, DrPH
Ohio	Thomas J. Halpin, MD, MPH	Gary D. Davidson, DrPH
Oklahoma	Paul Zenker, MD, MPH	Garry L. McKee, PhD
Oregon	David Fleming, MD	Charles D. Brokopp, DrPH
Pennsylvania	Dale R. Tavris, MD, MPH	Bruce Kieger, DrPH (Acting)
Rhode Island	Bela Matyas, MD, MPH	Raymond G. Lundgren, Jr., PhD
South Carolina	Dee Breeden, MD, MPH (Acting)	Harold Dowda, PhD
South Dakota	Kenneth A. Senger	Kathleen L. Meckstroth, DrPH
Tennessee	Robert H. Hutcheson, MD, MPH	Michael W. Kimberly, DrPH
Texas	Diane M. Simpson, MD, PhD	Charles E. Sweet, DrPH
Utah	Craig R. Nichols, MPA	A. Richard Melton, DrPH
Vermont	Robert Houseknecht, PhD	Burton W. Wilcke, Jr, PhD
Virginia	Grayson B. Miller, Jr, MD	D. B. Smit (Acting)
Washington	Mark Oberle, MD, MPH	Jon M. Counts, DrPH
West Virginia	Loretta E. Haddy, MA, MS	Frank W. Lambert, Jr, DrPH
Wisconsin	Jeffrey P. Davis, MD	Ronald H. Laessig, PhD
Wyoming	Stanley I. Music, MD, DTPH	Carl H. Blank, DrPH
American Samoa	Julia L. Lyons, MD, MPH	—
Federated States of Micronesia	Steven Auerbach, MD, MPH	—
Guam	Robert L. Haddock, DVM, MPH	Jeff Benjamin (Acting)
Marshall Islands	Tony de Brum	—
Northern Mariana Islands	Sean P. Flood, MD, MPH	—
Palau	Yugi Mesubed, MD	—
Puerto Rico	John V. Rullan, MD, MPH	Raul Baco Dapena, DVM
Virgin Islands	Alfred O. Heath, MD	Norbert Mantor, PhD

The *Morbidity and Mortality Weekly Report (MMWR)* Series is prepared by the Centers for Disease Control and Prevention (CDC) and is available on a paid subscription basis from the Superintendent of Documents, U.S. Government Printing Office, Washington, DC 20402; telephone (202) 783-3238.

The data in the weekly *MMWR* are provisional, based on weekly reports to CDC by state health departments. The reporting week concludes at close of business on Friday; compiled data on a national basis are officially released to the public on the succeeding Friday. Inquiries about the *MMWR* Series, including material to be considered for publication, should be directed to: Editor, *MMWR* Series, Mailstop C-08, Centers for Disease Control and Prevention, Atlanta, GA 30333; telephone (404) 332-4555.

☆U.S. Government Printing Office: 1993-733-131/67048 Region IV

DEPARTMENT OF
HEALTH AND HUMAN SERVICES
Public Health Service
Centers for Disease Control
and Prevention
Atlanta, Georgia 30333

FIRST-CLASS MAIL
POSTAGE & FEES PAID
PHS/CDC
Permit No. G-284

Official Business
Penalty for Private Use $300

HHS Publication No. (CDC) 93-8017